INTERLOBAR HYDROTHORAX IN CARDIAC FAILURE

BY

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Interlobar hydrothorax in cardiac failure is not common: White, August, and Michie (1947) did not encounter it in a series of 100 cases of hydrothorax in cardiac failure. The following case report illustrates the condition and the problems that may arise in diagnosis.

A male, aged 68, was referred to the Royal Infirmary, Edinburgh on November 19, 1948. For some months he had felt vaguely unwell with loss of appetite, but without any complaint referable to the cardiovascular or respiratory systems. His past health had always been good, except for an attack of jaundice two years previously, diagnosed as infective hepatitis. Clinical examination was normal. A barium meal showed no abnormality, but during screening the radiologist observed an opacity in the chest. Because of this, a chest X-ray was taken and showed (Fig. 1) an effusion between the upper and middle lobes on the right side; the cardiac shadow was seen to be enlarged.

Despite the negative chest history, the possibility of an interlobar empyema was considered, but diagnostic aspiration revealed a clear fluid which was examined with the following results: sterile on culture; no clot on standing; protein 1·25 g. per 100 ml.; and cell content, 100 per cu. mm. with 70 per cent endothelial cells and 30 per cent small lymphocytes. The fluid was considered to be a transudate of unknown ætiology. No treatment was prescribed.

The patient reported on December 10, 1948. He now complained of dyspnœa on exertion. Clinical examination showed bilateral basal crepitations which had not been present previously; the cardiovascular system was again found to be normal (no clinical evidence of the cardiac enlargement known to be present could be found). An X-ray of chest (Fig. 2) showed a considerable increase in the interlobar effusion with small additional effusions in both costophrenic angles and general vascular congestion in the lungs.

A tentative diagnosis of early congestive cardiac failure was made. The patient was asked to rest in bed at home.

He reported on January 11, 1949. The dyspnœa was now more troublesome. Clinical examination revealed deterioration in his condition. The pulse was irregular; the neck veins were distended; the liver was palpable two inches below the costal margin; there was pitting œdema of the ankles and some ascites. There was now no doubt about the presence of congestive heart failure. The urine contained no sugar or albumin. An X-ray of chest showed no change from Fig. 2. An electrocardiogram showed slow auricular fibrillation with evidences of an old apical infarct. Estimation of plasma proteins gave albumin 3·9 and globulin 2·0 g. per 100 ml.

A diagnosis of congestive cardiac failure secondary to old myocardial infarction and fibrosis was made. Treatment in hospital with digitalis and mersalyl resulted in rapid improvement. An X-ray of chest three weeks later showed disappearance of the fluid in the costo-phrenic angles and lessening of the interlobar effusion. After the initial rapid improvement progress was slow, and some œdema of the ankles and enlargement of the liver persisted despite intensive therapy. These features were still present when he was discharged two months later. Administration of digitalis and mersalyl was continued at home.

He reported again in November, 1949. He was now symptom free and had no signs of cardiac failure although slow auricular fibrillation was still present. An X-ray of chest showed that the interlobar effusion had disappeared, leaving some thickening of the interlobar fissure; cardiac enlargement was still present.

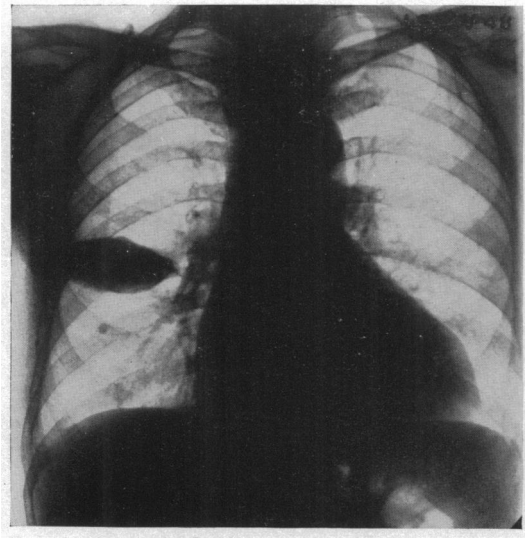

FIG. 1.—Shows a moderate-sized interlobar hydrothorax and a little enlargement of the heart without much general pulmonary congestion, before general signs of congestive failure had developed, November 19, 1948.

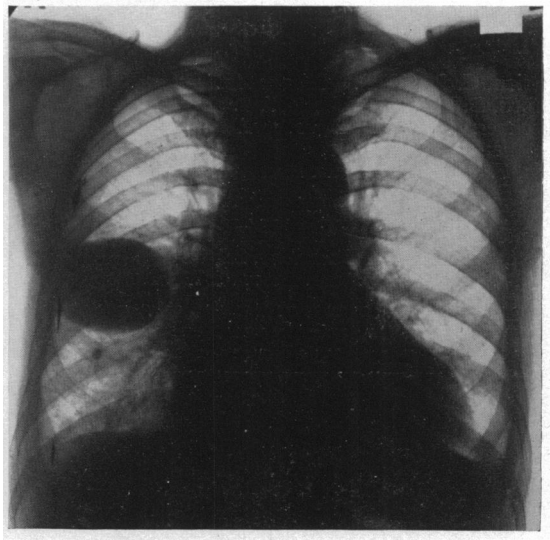

FIG. 2.—Shows an increase in size of the interlobar hydrothorax and of the pulmonary congestion when signs of congestive failure had become more evident, December 10, 1948.

Discussion

Including the case described above, 36 cases of cardiac interlobar hydrothorax have been reported —Helm (1917), Fleischner (1926), Stewart (1928), Kiser (1929), Freedman (1931), Steele (1932), Vesell (1932), Austrian (1932), Stein and Schwedel (1934), Shiflett (1935), Levitin (1937), Bedford and Lovibond (1941), Russakoff and Weinberg (1944). In 24 of the 29 cases in which the site was given, the interlobar fluid was in the right lesser fissure. In 27 of the 36 cases, the ætiology of the cardiac failure was stated and was in all a condition causing either left-sided or simultaneous left- and right-sided failure, namely aortic valve disease, hypertension, coronary occlusion, and myocardial fibrosis. In accordance with such ætiology, the age incidence is high, 23 of 28 cases being over 40 years.

In 14 of the 36 cases, there was no accompanying effusion in the general pleural cavities. In such circumstances, the correct diagnosis may not be thought of at first because of its rarity. Tuberculous loculated pleural effusion, loculated empyema, or even neoplasm may be wrongly diagnosed. A patient with coronary occlusion, which is a common cause of cardiac interlobar hydrothorax, may have fever, cough, chest pain, and leucocytosis, all of which may suggest empyema.

The cardiac interlobar hydrothorax, however, usually occurs in patients over 40 years of age, when tuberculous effusions are uncommon. The fluid, on examination, has the characteristics of a transudate. A cardiac condition causing left- or simultaneous left- and right-sided failure is present. Radiologically, the fluid is in most cases localized in the right lesser fissure and tends to disappear with treatment of the cardiac failure and to re-appear with relapse; the lung fields tend to show hilar and basal congestion due to associated pulmonary œdema.

Necropsy in some cases of cardiac interlobar hydrothorax—Helm (1917), Steele (1932), Austrian (1932), Levitin (1937), Russakoff and Weinberg (1944)—has shown that fibrous obliteration of the general pleural cavities has left no space other than an interlobar fissure in which the fluid could accumulate.

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